



## **FEATURES OF THE COURSE OF INFECTIVE ENDOCARDITIS IN AN ANIMAL EXPERIMENT**

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### **Abstract**

Infective endocarditis (IE) is a disease characterized by a severe course, the development of systemic complications, and an unfavorable prognosis, and it remains highly relevant in modern clinical practice. The creation of experimental models of infective endocarditis in animals makes it possible to study endothelial damage, bacteremia, vegetation formation, and the development of immune complex complications. The aim of this study was to evaluate the characteristics of the course of primary and secondary infective endocarditis in rats and rabbits using clinical-laboratory, instrumental, and pathomorphological assessments. During 2021–2022, 40 laboratory animals (20 rats and 20 rabbits) were examined under vivarium conditions. The results showed that primary infective endocarditis is more often characterized by an acute course, whereas the secondary form predominantly has a subacute course and is accompanied by immune complications (nephritis, vasculitis, synovitis). The obtained data confirm the correspondence of the experimental model with clinical observations and substantiate its significance for studying the pathogenesis of infective endocarditis and evaluating new treatment approaches.

**Keywords:** Infective endocarditis, experimental model, rat, rabbit, primary endocarditis, secondary endocarditis, vegetations, bacteremia, complications, nephritis, vasculitis, echocardiography.

## **Introduction**

Infective endocarditis is an infectious-inflammatory lesion of the cardiac endocardium and valvular apparatus, accompanied by a systemic inflammatory response as well as thromboembolic and immune complex manifestations. Despite advances in antibacterial therapy and cardiac surgery, infective endocarditis remains a disease associated with a high risk of mortality and disability [1–3].

A significant proportion of complications in infective endocarditis are caused not only by direct damage to the heart valves but also by immunopathological mechanisms, including nephritis, vasculitis, and joint involvement. This circumstance increases the importance of experimental models for a deeper understanding of disease pathogenesis [4–6].

Experimental models developed in rats and rabbits are widely used to study vegetation formation, embolic complications, and internal organ damage, since infective endocarditis in many cases is associated with primary endothelial injury of the valves followed by bacteremia [7–9].

## **MAIN PART**

In clinical practice, infective endocarditis is divided into primary (developing on intact valves) and secondary forms (occurring in the presence of heart defects, a history of endocarditis, cardiac surgical interventions, or implanted devices). Primary infective endocarditis is typically characterized by an acute onset and severe intoxication, whereas the secondary form often has a subacute course with a predominance of immune complications [1,4,5].

Under experimental conditions, modeling of infective endocarditis is based on a combination of factors: damage to the valvular endothelium (catheterization or mechanical injury), bacteremia (introduction of microorganisms or creation of a septic focus), and conditions for the formation of thrombotic masses. In particular, rabbit models of staphylococcal endocarditis and vegetation development have been well studied [8,9].

## **METHODOLOGY (MATERIALS AND METHODS)**

The study was conducted in 2021–2022 in the vivarium of TashPMI. A total of 40 laboratory animals—20 rats and 20 rabbits—were included in the experiment. Experimental conditions corresponding to primary and secondary infective endocarditis were modeled in the animals. Inclusion criteria were a satisfactory baseline condition of the animals and the absence of signs of acute somatic disease.

The observation protocol included clinical and anamnestic assessment (body temperature, behavior, appetite, body weight), laboratory investigations (complete blood count and biochemical parameters), paraclinical methods (electrocardiography, echocardiography, chest radiography of the heart), and pathomorphological examination of the heart valves and myocardium.

Statistical analysis was performed using Statistica® 6.0 software. The significance of differences was evaluated using Student's *t*-test, and differences were considered statistically significant at  $p < 0.05$ .

## **ANALYSIS (RESULTS)**

In the experiment, primary infective endocarditis developed more frequently against the background of generalized infectious conditions and invasive interventions. Specifically, it was observed in 28% of cases in association with experimental sepsis, in 21% with purulent-inflammatory skin processes, in 14% after intravenous catheterization, and in 7% after modeled dental interventions.

Secondary infective endocarditis was mainly recorded in the presence of pre-existing cardiac pathology: in 34% of cases with congenital heart defects, in 20% as a complication of postpericardial syndrome, in 31% following previously induced experimental endocarditis, and in 15% after cardiac surgical interventions.

An acute course was observed in 71% of animals with primary endocarditis, whereas a subacute course predominated in 64% of cases of secondary endocarditis, which corresponds to clinical observation data [1,4].

The structure of complications included renal involvement (70–76%), splenomegaly (55%), arthralgia and arthritis (41%), myocarditis (25%), and cutaneous manifestations (5–8%). Pathomorphological examination revealed inflammatory changes of the valves, vegetations, microthromboembolism, and signs of vasculitis [4–6].

## **DISCUSSION (OUTCOME)**

The obtained results demonstrate that the experimental model of infective endocarditis in animals closely resembles the processes observed in clinical practice. The primary form is characterized by a rapid and severe course, while the secondary form develops more slowly and is accompanied by immune complications. The high frequency of renal and vascular damage confirms the significant role of immune mechanisms in the pathogenesis of infective endocarditis [1–3,5].

This model is suitable for assessing vegetation development, systemic complications, and the effectiveness of therapeutic interventions, and it has particular prospects for studying staphylococcal endocarditis in rabbits [8,9].

## **CONCLUSION**

According to the results of the experimental study, primary infective endocarditis in animals most often develops against the background of sepsis and catheterization, whereas the secondary form is mainly associated with heart defects and cardiac surgical interventions. Secondary infective endocarditis is characterized by a subacute course and a high frequency of immune complications. The infective endocarditis model developed in rats and rabbits is promising for in-depth investigation of disease pathogenesis and for evaluating new preventive and therapeutic approaches.

## **REFERENCES**

1. Delgado V., Marsan N.A., et al. ESC Guidelines for the management of infective endocarditis. *European Heart Journal*, 2023.
2. Baddour L.M., Wilson W.R., Bayer A.S., et al. Infective endocarditis in adults. *Circulation*, 2015.
3. Demin A.A., et al. Clinical guidelines for infective endocarditis. *Russian Journal of Cardiology*, 2021.
4. Lobzin Yu.V., et al. Clinical masks of infective endocarditis. *Journal of Infectology*, 2015.
5. Efremova O.A. Infective endocarditis (review). 2010.
6. Vardugina N.G., et al. Clinical and morphological aspects of infective endocarditis. 2016.



7. Shevchenko Yu.L. Infective Endocarditis: Pathogenesis, Diagnosis, Treatment. Monograph, 2015.
8. Wang M., et al. A rabbit model of staphylococcal infective endocarditis. 2013.
9. Kinney K.J., et al. Vegetation formation in infective endocarditis. 2022.